

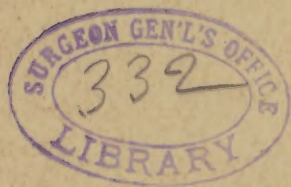
MACKENZIE, (J. N.)

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A Contribution to the Patho-
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and Chronic Coryza.

BY
JOHN N. MACKENZIE, M. D.,
BALTIMORE.

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A CONTRIBUTION TO THE PATHOLOGICAL HISTOLOGY OF ACUTE AND CHRONIC CORYZA.*

BY JOHN N. MACKENZIE, M. D.,
BALTIMORE.

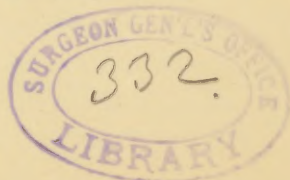
IN the "Philadelphia Medical News" of October 4, 1884,† I gave an account of the changes found in the different stages of intra-nasal inflammation, which was based upon the anatomical study of a large number of neoplasms removed from the nasal passages by means of the cold-wire snare. The object of the present communication is to call attention to some additional points in the pathological anatomy of that affection, which I trust may prove of general interest, both from a clinical and histological point of view.

The first specimen that I submit to your examination (Fig. 1) represents the microscopical appearances of a section through the inferior turbinated body of a man dead of Bright's disease, the result of long-standing mitral regurgitation and insufficiency.‡

* Read before the American Laryngological Association, June 25, 1885.

† "Some Notes on the Pathology of Intra-nasal Inflammation."

‡ I am indebted to Dr. W. T. Councilman, Associate in Pathology in the Johns Hopkins University, both for the beautiful section and



Among the ordinary visceral changes usually found in such cases there were traces of old infarctions in both kidneys and spleen. The nasal passages presented the appearances commonly observed in the condition known as coryza, and in the permanent puffy condition found in more chronic inflammation, the most noticeable being intense engorgement of the cavernous tissue, especially well marked over the lower half of the middle and posterior portion of the inferior turbinated body. Upon cutting into the engorged bodies with a scalpel, the blood, which flowed freely, could be squeezed from them as water from a sponge. The mucous membrane of the maxillary sinuses was not in the slightest degree tumefied, and presented simply a more or less cyanotic appearance.

Under the microscope, the condition which at once attracts the eye is the enormous dilatation of the erectile spaces and the extreme tenuity of the intercellular walls. Here and there rupture of the latter has occurred from the enormous blood pressure, and two or more spaces in this way communicate. Along the inner walls of the dilated spaces are seen congregations of lymphoid corpuscles, and in some of them collections of fibrinous exudation.

The mucous membrane proper presents no noticeable pathological change. The epithelial layer is intact. At some places it seems slightly thicker than at others, but, beyond a moderate amount of cellular infiltration of the tissues beneath the basement membrane, there is nothing in the mucous layers to call for special remark.

utting out from and attached to the wall of the central sinus (in the diagram) is a well-formed parietal thrombus, to which I would like to call particular attention.

notes of the post-mortem examination upon which these remarks are based, and for the accurate drawings which illustrate the text,

I bring this case before you for discussion as one of peculiar interest, for it is instructive in many ways. So far as I am aware, the histological appearances in acute coryza have never been investigated. In this specimen we have the probable microscopical picture of that

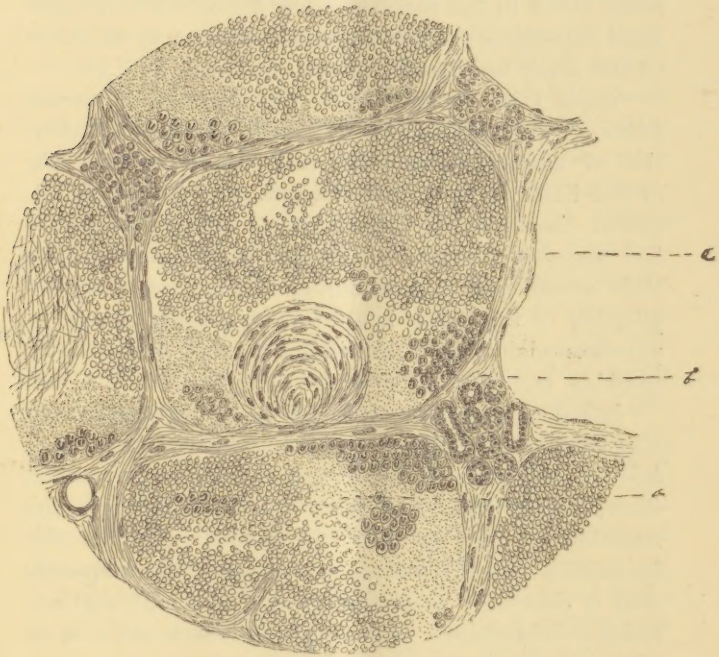


FIG. 1.

disease. Here, too, is illustrated the minute anatomy of the puffy condition or state of permanent dilatation of the erectile spaces which characterizes the second period of the simple inflammatory stage before hypertrophic changes

have developed, and to which I have called attention elsewhere.*

Of great interest, too, is the formation of the parietal thrombus, as illustrative of a mode of obliteration of the erectile spaces heretofore unrecognized.

In connection with the negative post-mortem appearances found in the maxillary sinus I would like to offer some suggestions on the relation of inflammatory affections of that cavity to intra-nasal processes.

While I am convinced that the chronicity of certain forms of nasal disease, the persistency of a fair proportion of neuralgic (and other reflex neuroses) affections of the head and face, are traceable to latent or unsuspected chronic antral inflammation, while it is doubtless true that affections of the maxillary sinus often run their course unrecognized during life, the *secondary* implication of that cavity in acute and chronic inflammatory processes originating in the nasal passages is, comparatively speaking, rare.

Inflammation of the maxillary sinus may result from congenital or acquired defect of the naso-antral aperture, or from its closure by the swollen and hypertrophied nasal tissue, polypi, crusts, etc., conditions which may favor the retention and decomposition of the antral secretion. Inflammation from this cause, however, occurs less frequently than is universally taught by inference from purely theoretical considerations. I base this assertion not only on clinical observation, but also on the results of post-mortem investigation. Those of you who choose to investigate the anatomical aspect of this question will be amazed to find how infrequently the mucous covering of the sinus partici-

* See article referred to, and also a paper in the "Medical News" of April 4, 1885, entitled, "Notes on the Classification, Diagnosis, and Treatment of the Stages of Chronic Nasal Inflammation."

pates in the inflammatory condition of the nasal fossæ. It is the rule to find the most marked hypertrophy of the nasal tissues, and even complete atrophy, associated with a normal condition of the sinus. What is the explanation of this fact?

On the nasal aspect of the naso-antral opening is a duplicature of the erectile tissue of the turbinated bodies, which in all probability serves the useful purpose of excluding from the cavity of the sinus, by means of the anatomical barrier involved in its erection, various irritants derived from the external world and nasal passages. This tissue ceases abruptly at the antral aperture. On the other side of the opening lies the peculiar loose, vascular membrane of the sinus. In the majority of cases this zone of erectile tissue constitutes the extreme anatomical limit of the nasal inflammation. The thin, loose, serous-looking membrane of the sinus differs essentially from the nasal mucous membrane, and especially that portion which surrounds the entrance into the maxillary sinus, and the abrupt transition of the one tissue into the other would therefore militate, on theoretical grounds, against extension of the inflammatory process into the antrum. In other words, I believe that the so-called extension of inflammatory action from the nasal passages to the antrum—the secondary inflammatory implication of that cavity—is of comparatively infrequent occurrence; and that, when inflammation of the sinus from extension is assumed, the condition will be found, on post-mortem examination, to be one of simple congestion, with possibly some accumulation and retention of the antral secretion from mechanical causes. Even if the amount of secretion be temporarily increased, the great power of absorption possessed by the membrane of the sinus will usually suffice to make it a matter of secondary importance.

I am inclined to believe that too much stress is laid upon the decomposition of antral secretion as a factor in the production of inflammation. Take, for example, a simple coryza. With the general erection of the turbinated tissues the orifice of the antrum is completely closed, congestion follows, with possibly an increased secretion of the antral fluid. Whether this secretion, thus hermetically sealed, so to speak, in the antrum undergoes decomposition before the patency of the opening is restored and the air gains admittance, is questionable; but, on the other hand, that decomposition does occur is beyond dispute, for, when the swelling from the acute rhinitis subsides, the aperture becomes free and, in some instances, the fœtid antral secretion is discharged into the nasal passages, a fact which accounts for the peculiar factor of the expired air from the nostrils which is sometimes observed at the close of a severe coryza.

In the second specimen (Fig. 2) are shown the microscopical appearances in the hypertrophic stage of chronic nasal inflammation before complete obliteration of the erectile spaces has taken place. Contrast the condition here with that found in the first specimen and in the acute and earlier stage of chronic rhinitis. Observe closely the little buds or bulbous processes which jut out from the walls of the cavernous sinuses, and which are made up of outgrowths from the newly formed intercellular connective tissue. In my anatomical study of this stage I overlooked these little buds, and my attention was subsequently called to them by my friend, Dr. Councilman, who had taken one of my sections for examination.

Projecting inward, they form bulbous processes or septa of varying thickness, which sometimes interlace, sometimes form bands which connect one portion of the sinus-wall with another, thus constituting another

factor in the division and obliteration of the cavernous spaces.

Care should be taken not to confound them with the remains of a thinned and ruptured intercellular wall; in the latter case a similar projection will usually be found at a

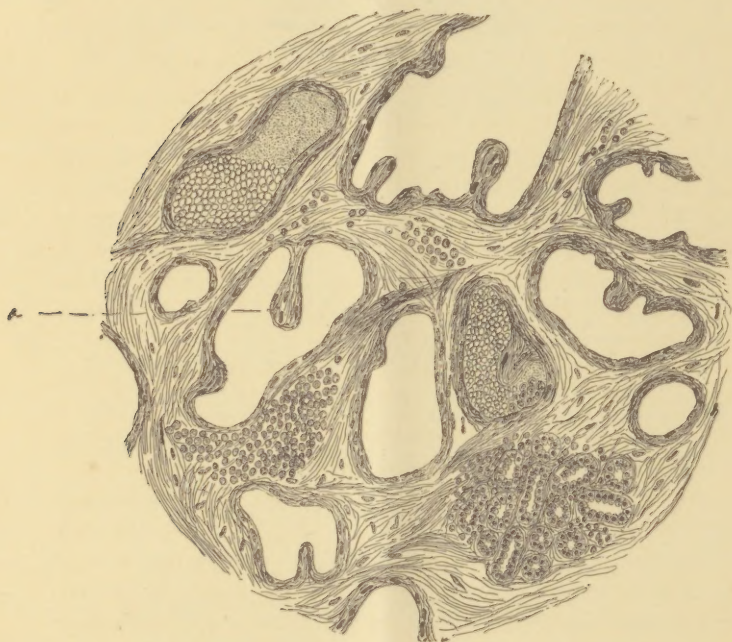


FIG. 2.

point diametrically opposite to that occupied by the projecting band.

We have, then, four modes of obliteration of the erectile spaces in nasal inflammation:

1. By the contraction of the newly formed intercellular fibrous bands,

2. By obliteration of their lumen by masses of round cells which resemble the white corpuscular elements of the blood. (See first article in the "Medical News.")

3. By the formation of thrombi in certain cases.

4. By the process of septa formation.

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